

# Syncope in a 67-year-old man

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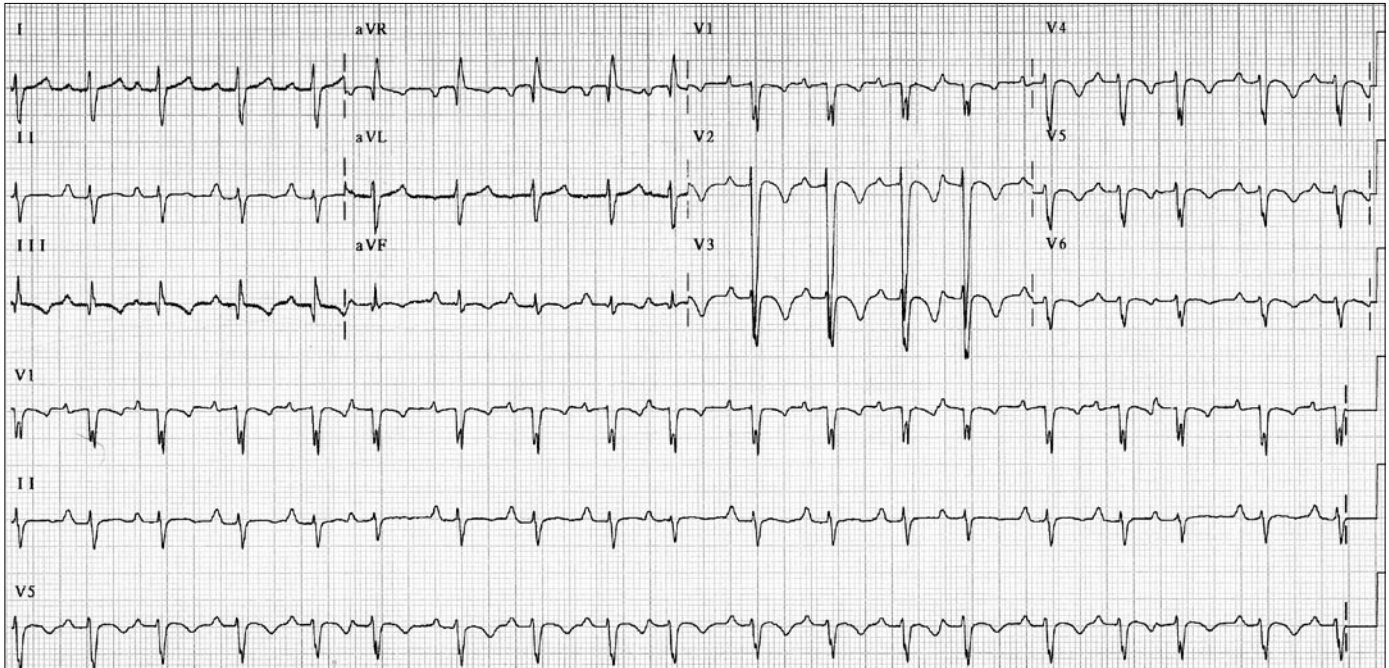


Figure. Electrocardiogram performed in the emergency department. See text for explication.

**A** 67-year-old man with an unremarkable medical history except for high blood pressure came to the emergency department because of a syncopal episode. He denied dyspnea and chest pain. An electrocardiogram was recorded (Figure).

The rhythm on the electrocardiogram is sinus tachycardia with frequent premature atrial complexes—the 2nd, 5th, 9th, 13th, and 16th P waves. A P-wave axis of  $+70^\circ$  and slightly tall P waves in leads II (2.5 mm) and  $V_2$  (1.5 mm) suggest the possibility of right atrial enlargement. The QRS axis is markedly rightward ( $+175^\circ$ ), and the QRS duration is normal (0.09 seconds). Precordial R-wave development is markedly delayed, a so-called clockwise rotation of the QRS complex, with the S wave larger than the R wave in each of the precordial leads. Inverted T waves are found across the precordium and in leads III and aVF, where they are accompanied by small q waves; lead II shows neither q's nor inverted T's. All of these findings suggest acute cor pulmonale, which is usually due to pulmonary embolism (1–4).

One of the findings in this electrocardiogram is the  $S_1Q_{III}T_{III}$  pattern described 70 years ago by McGinn and White, the first account of electrocardiographic changes in pulmonary embolism

(5). This electrocardiogram calls to mind infarction, injury, and/or ischemia both *inferiorly* and *anteriorly*, which, as emphasized by Marriott, also suggests pulmonary embolism (2, 6). In addition to the changes demonstrated in this electrocardiogram, pulmonary embolism may cause atrial flutter or fibrillation, right ventricular premature beats, ventricular fibrillation, right bundle branch block (complete or incomplete) or less marked right ventricular conduction delay, right ventricular hypertrophy, ST-segment elevation, a long QT interval, and pulseless electrical activity (1–7) (Table).

The presence and number of these electrocardiographic findings in a patient with pulmonary embolism depend on how much of the cross-sectional area of the pulmonary arteries is occluded and on the time course of the disease (1, 5). Sinus tachycardia is the most common finding but is nonspecific. In fact, no single

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**Table. Electrocardiographic abnormalities in pulmonary embolism\***

Rhythm	Sinus tachycardia+ Atrial premature complexes+ Atrial flutter Atrial fibrillation Right ventricular premature complexes Ventricular fibrillation Pulseless electrical activity Sinus bradycardia or asystole (rarely)
P waves	Rightward axis ( $\geq 75^\circ$ )± Tall ( $>2.5$ mm) in leads II, III, or aVF±
QRS complex	Right axis deviation or rightward axis shift+ Clockwise rotation+ Right ventricular conduction delay Right ventricular hypertrophy Pseudoinfarction Inferior+ Anterior Both Left axis deviation (rarely)
ST segment	Elevation inferiorly and/or anteriorly Depression
T wave	Inversion inferiorly+ Inversion anteriorly+ QT prolongation

+ indicates present on current electrocardiogram; ±, borderline finding on current electrocardiogram.

\*Modified from reference 1.

finding is specific, and the diagnosis of pulmonary embolism is suspected on the basis of the number and severity of the changes and especially on the clinical setting (1, 4, 5). Allen and Surawicz found that the electrocardiogram provided diagnostic support in 69% of patients in whom the physician suspected pulmonary embolism; however, pulmonary embolism was confirmed in only 8% of patients in whom the electrocardiographer suggested the diagnosis to the clinician (4).

In our patient the diagnosis was confirmed by a ventilation-perfusion scan that showed normal ventilation and multiple segmental perfusion defects in each lung. As would be expected from the presentation with syncope, always an ominous symptom indicating extensive pulmonary embolization, and the striking electrocardiographic changes, the echocardiogram demonstrated right atrial dilatation, right ventricular dilatation and hypokinesis, and a flattened ventricular septum during diastole. Serum troponin I was minimally elevated (0.12 ng/mL; normal  $<0.05$ ).

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